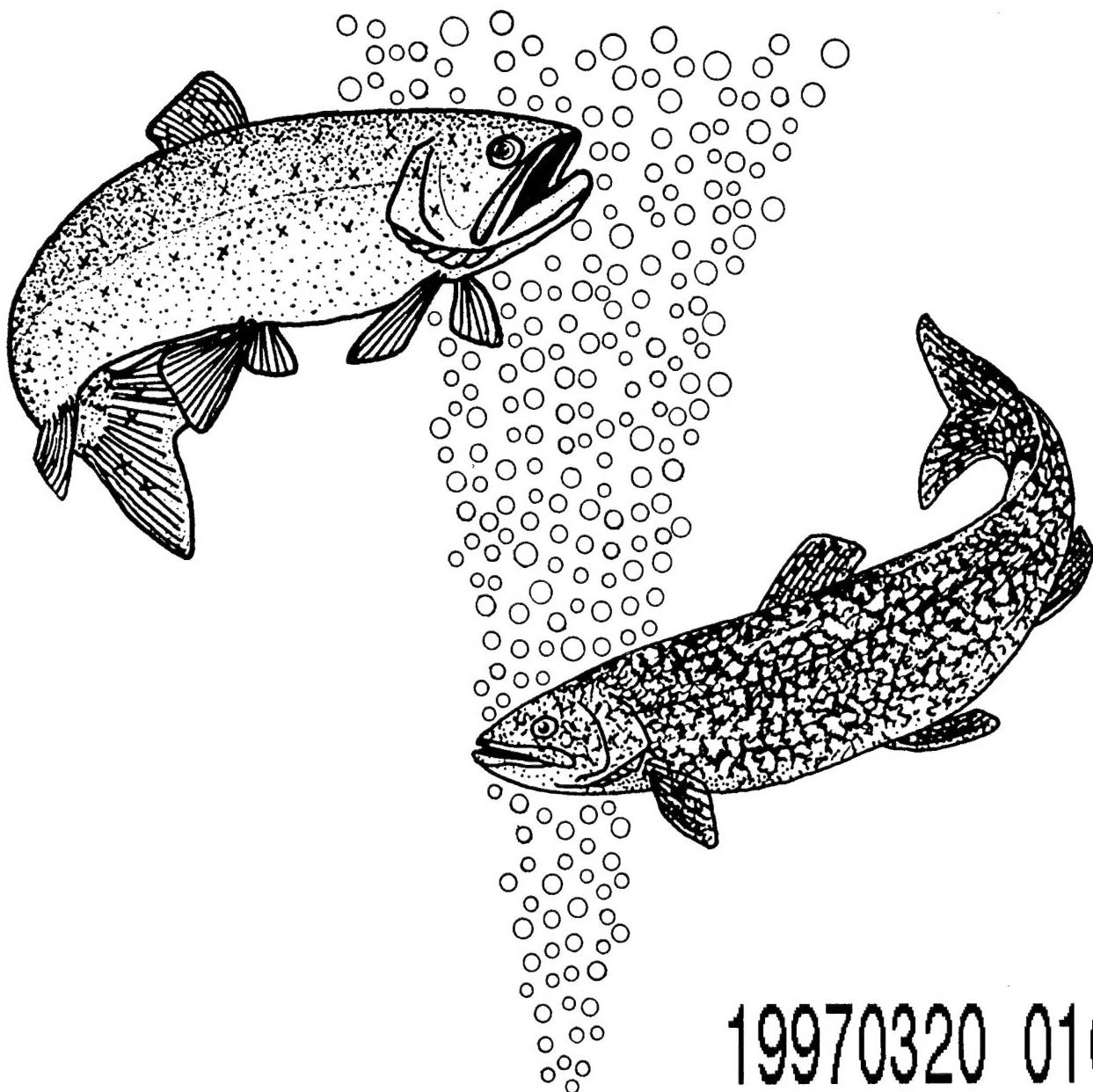


Gas Supersaturation in Fisheries: Causes, Concerns, and Cures



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Cover: Atlantic salmon (above) and lake trout (below), two species highly sensitive to gas bubble disease, thrive in water treated by oxygen injection. (Drawing by S. M. Jeffrey)

Gas Supersaturation in Fisheries: Causes, Concerns, and Cures

by

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Introduction

Gas embolisms in fishes have been recognized as a problem since at least 1670, and the term "supersaturation" was used as early as 1866 to describe an excess of any gas in solution. Studies carried out near the turn of the century suggested that fish culturists understood some of the problems involved in "gas bubble disease," and knew how to apply certain preventive techniques.

Gas bubble disease was first described by Gorham (1901) as consisting of "vesicles of gas invading all the superficial parts of the fish, especially fins, eyeballs, and in loose connective tissue of the orbits, so that the eyes were forced from their sockets; less commonly bubbles formed beneath the lining of the mouth, in the gill arches, or beneath the skin, so that scales were raised from the surface. The swimming behavior of the fish was disturbed, especially in maintenance of horizontal equilibrium."

Gorham was perhaps the first investigator to relate gas bubble disease problems to a reduction in partial pressures rather than to a pathogen. He calculated that, in a fish raised from a depth of 300 fathoms to the surface, the contained gases expanded to more than 54 times the original volume. He noted that fish taken from great depths were usually dead when they reached the surface, the eyes were protruded, the air bladder was greatly distended or ruptured, and the gastrointestinal tract was often everted from the mouth or anus. In fish less severely affected by supersaturated water, reentry into deep water alleviated the problem because both pressure and gas solubility increase with depth.

Gas bubble disease in aquatic animals (which is analogous to the bends in humans) was first observed and recognized in fishes held in seawater in a public aquarium at the Bureau of Fisheries station at Woods Hole, Massachusetts, in about 1900 (Marsh 1903; Marsh and Gorham 1905). The signs of the problem were recognized, as well as the likelihood of mortality if excess air remained in the bloodstream. Nitrogen excess was considered more important than oxygen excess in causing the disease, and it was known that nitrogen excess could be the sole cause. Marsh and Gorham (1905) suggested that supersaturation could be corrected by deaeration, which occurred spontaneously if water was allowed to stand, or more quickly if the surface area exposed to the atmosphere was increased.

The mystery of gas bubble disease at Woods Hole was solved when Marsh (1903) recognized that water flowing to the laboratory aquaria was pressurized, and that bubbles of air that were sucked in through leaks in the intake pipe were forced into solution by the pressure pump on the water supply line. The supersaturated water was so lethal that new display fish had to be introduced into the aquarium every few days; sometimes more than half the fish died within 48 h. He later demonstrated that nitrogen gas decreased to nontoxic levels and oxygen increased when the water flow was subdivided and diverted through a simple aeration device consisting of five or six perforated dishpans (Marsh 1910). Such an apparatus aerates water if it is deficient in dissolved oxygen and deaerates it if dissolved gases are excessive.

Various aspects of gas supersaturation and gas bubble disease were reviewed by Rucker (1972), Harvey

(1975), Weitkamp and Katz (1980), Speece (1981), and Colt (1986).

Natural Occurrences of Supersaturated Water

Groundwater has long been known to differ in the variety and concentration of dissolved gases. Marsh (1910) observed that water from freshwater springs and wells is often supersaturated, and this phenomenon has been reported at many fish hatcheries throughout the country that draw their water supplies from wells or springs. Nitrogen concentrations in the water from 15 artesian wells and 2 springs ranged from 118% to 159%, and saturation in most exceeded 140% (Harvey 1975). Well water at both the Erwin (Tennessee) and Nashua (New Hampshire) National Fish Hatcheries contains an excess of nitrogen that is slowly lethal to fish. The supersaturation is believed to be a result of either high pressures that prevail in some underground aquifers or increases in temperature as the water comes to the surface, or of a combination of the two factors.

As a result of the agitation and turbulence caused by waterfalls in streams, water traps air bubbles and often transports them to considerable depths. As depth and pressure increase, the bubbles (mainly nitrogen and oxygen) become dissolved in water. Air is entrained (trapped) whenever air and water are in contact at pressures greater than the ambient atmospheric pressure. Transfer rates increase with depth because the pressure increases. Saturation produced by waterfalls may range from slight to about 134%; however, relatively high values have been reported in rivers without waterfalls (Harvey 1975).

Photosynthesis by algae during heavy algal blooms has produced gas saturation of 327% in fresh water and 250% in salt water; affected fish showed the characteristic symptoms of gas bubble disease, and death usually resulted from blockage of blood circulation in the gills by the gas bubbles (Harvey 1975). Although supersaturation of oxygen was implicated in the reported fish kills, other gases may have been present; for example, nitrogen gas was at 120% of saturation in the salt water in which total gas saturation was 250%.

Ice formation sometimes produces a physical barrier that prevents the release of dissolved gases when water is converted to ice (Colt 1986). As ice forms, dissolved gases come out of solution; if they cannot be released to the atmosphere, they may become concentrated in the remaining water and cause supersaturation.

Artificial Occurrences of Supersaturated Water

Physical factors related to a water system can also lead to gas supersaturation problems. The leaky suction pipe at Woods Hole in 1900 (described previously) produced supersaturation in water that originally was in complete equilibrium with the air (Harvey 1975). If air is sucked into water through pumps, pipes, or turbines under pressure, supersaturation with nitrogen increases much more rapidly than supersaturation with oxygen because of the 80:20 ratio of the two gases in the air—even though solubility in water is greater for oxygen than for nitrogen. Many fish culture facilities cite artificially induced supersaturation of water supplies as the cause of gas bubble disease.

Water sometimes becomes supersaturated at hydroelectric installations when river water falls over spillways. Problems caused by the hydropower dams in the Columbia River are perhaps the best known. Nitrogen gas saturation levels at 26 locations there ranged as high as 140% (Harvey 1975), and the supersaturation developed at dams persisted to some degree throughout the length of the reservoirs formed by the next dam downstream. Lethal nitrogen gas supersaturation was demonstrated by holding young coho salmon (*Oncorhynchus kisutch*) and chinook salmon (*O. tshawytscha*) in cages at four depths. Only fish held in water deeper than 2.5 m avoided gas bubble disease. Elsewhere it was shown that survival increased with increasing depth in juvenile sea-run rainbow trout (steelhead), *Salmo gairdneri*, suspended in cages at various depths in water supersaturated with air (Knittel et al. 1980).

Gas supersaturation problems in the Columbia River prompted numerous investigations by concerned fishery managers, fishery researchers, and agencies such as the U.S. Army Corps of Engineers and the U.S. Environmental Protection Agency; the problem was described by Ebel (1969), Beiningen and Ebel (1970), and Roesner and Norton (1971). Gas bubble disease in the Columbia River was eventually alleviated by altering the design of dams to eliminate deep plunges of water into spilling basins.

An increase in water temperature can also cause nitrogen supersaturation, even if the water is at air equilibrium—that is, 100% saturated with nitrogen—before warming begins. Each rise of 1 Celsius degree in water temperature increases saturation by about 2%. The heat may originate from cooling towers of industrial plants, from the mixing of warm water with cold water, or from the intentional heating of fish culture water to stimulate

fish growth. Gas bubble disease has sometimes developed when the water temperature increased by only 3 to 5 Celsius degrees.

Other mechanisms for the production of gas supersaturation and for natural occurrences of gas supersaturation, mentioned by Colt (1986), included bacterial action and physiological processes.

Signs of Gas Bubble Disease

A classical description of the development, signs, and characteristics of gas bubble disease was given by Marsh and Gorham (1905), who conducted analyses for oxygen, nitrogen, and carbon dioxide in culture water and analyzed the gases inside bubbles. Gas bubbles are basically a precipitate formed from gases within the water itself. They tend to collect on the surfaces of container walls or fish. In highly supersaturated water, bubbles collect on the external surfaces of fish within 3 min and increase in size and envelop a fish completely within 10 min. After 24 to 48 h, intracellular bubbles may produce lesions due to the inflation of mucous membranes that line the mouth cavity (Fig. 1), the skin, and especially the fins. Inflation of the membranes behind or within the eyeballs produces a conspicuous clinical sign known as "popeye" (Fig. 2). These external characteristics are only overt signs of gas bubble disease; they may lead to loss of equilibrium, but not usually to death. The gross appearance of the external gaseous lesions is often more or less peculiar to the species.

Internal bubbles cause death. Osmotic pressures on both sides of the gill membrane tend to equalize and, because blood and water have nearly the same saturation point, the gas excesses are about equal in the bloodstream and water. As a consequence, any supersaturation in the water is readily transferred into the bloodstream of the fish. Blood vessels may contain various quantities of free gas, ranging from a few small bubbles scattered through the vessels to larger bubbles that, for example, distend the bulbous of the heart to several times its normal size. The auricle may continue to beat, but propels no blood. The branchial artery or ventral aorta is often free of blood and turgid because of the gas pressure. The most common and most significant lesions are generally in the gill filaments. Gas bubbles plug the filaments, even when the presence of gas is not evident in the rest of the body.

The cause of death in gas bubble disease is usually asphyxiation, caused by gas emboli in the gill filaments or heart, or both. Nitrogen is chiefly, if not solely, responsible for gas bubble disease in fish. Samples of bubbles from the hearts of various fishes have indicated that the gas that causes fatal emboli in the vessels is almost pure nitrogen. Fish that die of gas bubble disease rapidly lose the overt evidence of gas bubbles and generally no external signs remain after 24 h. This disappearance of signs often complicates the diagnosis of gas bubble disease.

Recent investigators have failed to provide a more thorough description of processes involved in gas bubble disease, but Bouck (1980) described clinical signs

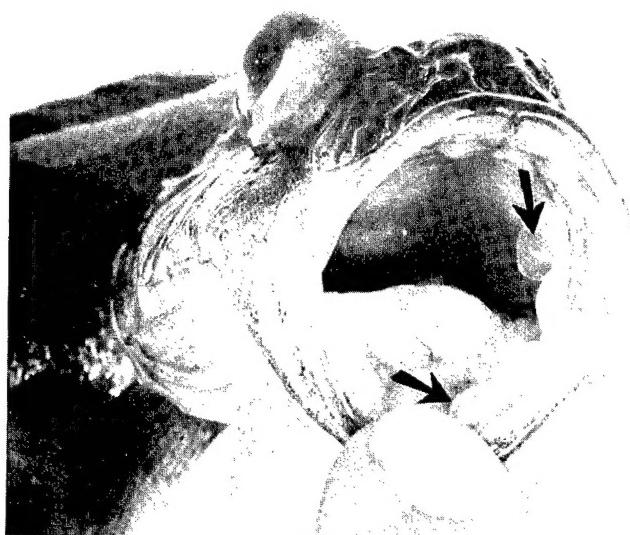


Fig. 1 Gas bubbles (arrows) in the mouth of a yellow perch (*Perca flavescens*) that was held in gas-supersaturated water for 6 weeks. Other signs of distress were loss of stamina for feeding, loss of appetite, frequent surfacing in attempts to maintain equilibrium, and eventually morbidity.



Fig. 2. Yellow perch, showing characteristic external signs of gas bubble disease. Gas bubbles are noticeable within the eye integument (arrows, *upper view*) and popeye is illustrated in the *lower view*.

associated with the disease. As the small bubbles formed in the bloodstream pass through the blood vessels and heart, the fish often show signs of increased irritation, possibly in reaction to pain receptors in blood vessels of the gills; they become restless or erratic and avoidance responses may increase. As the emboli enlarge, activity is often reduced; however, fish at this stage can still recover. The next stage produces mortalities triggered by stoppages of blood flow that are caused by emboli that fill the heart and the ventral aorta from the capillary bed of the gills back to the heart. Emboli prob-

ably cause a variety of other dysfunctions in the cardiovascular system and in various body organs. Chronic exposure to supersaturation leads to the development of emphysemas—sometimes grotesque in size and proportions—in a large number of cavities. Exophthalmia (popeye) also may occur, and lead to the loss of the lens or of the entire eyeball. Even so, fish often continue to feed and appear to be functional. Emboli and the other signs of emphysema disappear after death because the heart ceases to supply the gases needed for continued inflation.

Toxic Levels of Supersaturation

Gas supersaturation has caused fish mortalities under a wide variety of cultural and natural conditions. Supersaturation of 139% below the Harry S. Truman Dam in the upper Osage River, Missouri, killed nearly 500,000 fish in the spring of 1978 and 1979 (Crunkilton et al. 1980). In the laboratory, the 96-h median lethal concentration (LC₅₀) was 140% for speckled dace, *Rhinichthys osculus*, and 119% for cutthroat trout, *Salmo clarki* (Nebeker et al. 1980). Estimated threshold concentrations for these species were 123% and 114%, respectively. In salmonids, nitrogen supersaturation should not exceed 103% for eggs, fry, and fingerlings, or 105% for older fish (Owsley 1981). Gas emboli were reported in the gills of dying hatchery trout at 105% saturation (Bouck 1980). Stresses associated with low levels of gas supersaturation may not produce mortality directly, but the susceptibility of affected fish to secondary infections by pathogenic organisms generally increases.

Pacific salmon and rainbow trout generally avoided 145% and 125% saturation but did not avoid 115% (Stevens et al. 1980). However, many steelhead died of gas bubble disease because they did not always avoid highly supersaturated water. The ability to detect and avoid supersaturated water seems to vary with the species of fish and with water depth and temperature.

The general criterion of 110% for gas supersaturation of water, established by the U.S. Environmental Protection Agency, does not reflect the variation in sensitivity among species of fish. The 110% criterion has been disputed as being too high for fish hatcheries and too low for rivers deeper than 1 m (Ebel et al. 1979); 115% has been suggested as being a more acceptable limit for deep rivers. On the other hand, some hatchery managers have reported production problems at 104% and 105% saturation in the culture of especially sensitive fishes such as Atlantic salmon (*Salmo salar*) and lake trout (*Salvelinus namaycush*).

Fish in hatchery systems are more vulnerable than wild fish to the effects of supersaturation because they are generally confined in shallow water. Hatchery fish are also subjected to stresses of confinement and culture that may be aggravated by the added stress caused by supersaturation. Supersaturation in groundwater is generally seasonal, but peak concentrations and durations are unpredictable. Hatchery managers must be aware of this variability, and be prepared to treat water supplies on an emergency basis or continuously.

Rationale of Gas Supersaturation

The dissolved gas pressure in water is normally equal to atmospheric (barometric) pressure. This balance can be upset under certain natural or man-made conditions. The difference between total gas pressure and local barometric pressure (called the differential pressure, and indicated by the symbol ΔP) can be measured directly with membrane diffusion instruments. Differential pressure is sometimes preferred over percent supersaturation for reporting results (Colt 1984). If ΔP is less than zero, bubbles cannot form, regardless of the degree of supersaturation of a single gas. The value of ΔP can also be reported as a percentage of the local barometric pressure.

The basic formula for calculating gas supersaturation is

$$TGP(\%) = \frac{BP + AP}{BP} \times 100$$

where TGP = total gas pressure, BP = barometric pressure, and AP = atmospheric pressure. Detailed information on calculations was given by Colt (1984) and Dawson (1986).

Detection and Measurement of Gas Supersaturation

Gas supersaturation is often first detected when the signs of gas bubble disease are seen in fish. These signs appear too late, however; heavy mortality may occur before equipment and procedures can be installed to alleviate the problem. A more practical approach is the use of an instrument such as a satrometer to test and monitor the water supply for possible gas supersaturation. The preferred method of analysis for relative gas saturation is direct measurement of the total gas pressure with membrane-diffusion instruments such as those described by Fickeisen et al. (1975) and Bouck (1982). Routine monitoring should be done at least weekly at production or research facilities to detect supersaturation that might develop from air leaks in suction pipes or shaft seals. Continuous monitoring may be desirable if gas saturation varies more than 2% from day to day, or if treatment equipment is to begin automatically whenever a preselected level of supersaturation occurs.

A computer program that efficiently performs all of the calculations needed to determine gas pressures and percent saturation values for water was developed by

Dawson (1986). Measurements of total gas pressure, dissolved oxygen, temperature, and barometric pressure are used to compute the percent saturation of total gases, oxygen, and nitrogen plus argon, according to the methods of Weiss (1970). A complete record can be made available in a few seconds, without the tedium or potential errors associated with manual calculations.

In a recent evaluation of gas bubble disease, Colt (1986) described the problem as gas bubble trauma rather than as a disease. He discussed research that strongly supports the concept that gas bubble trauma may be either acute or chronic—acute if gas supersaturation is high and chronic if it is low; however, he did not clearly differentiate between high and low levels. Because the problem is not caused by a pathogen and is not communicable, the term gas bubble trauma may be the more appropriate because it more accurately describes the stresses or physical injury associated with the disorder.

Treatment of Gas Supersaturation in Water

The earliest recorded technique for the treatment of gas supersaturation problems was the use by Marsh (1910) of perforated aeration pans mounted one above the other. Pebbles were added to the water intake troughs to increase the surface area and decrease gas pressure. In other early efforts, as reviewed by Harvey (1975), hatchery aerators were devised in 1936 that passed water over an inverted cone to increase the water surface area, and in 1948 it was cascaded from trough to trough—procedures that reduced nitrogen saturation from 140% to about 104%. In 1953, the passage of spring water over a series of three weirs reportedly decreased the nitrogen content to 110%, and in 1962 the use of a splash tower with 12 sets of baffles reduced nitrogen saturation to 102%. None of these attempts effectively eliminated gas supersaturation in water, but most reduced supersaturation to levels tolerated by most species.

Recent attempts have involved more sophisticated aeration devices, such as splash plates, plunges, spray nozzles, screens, perforated plates and buckets, mechanical aerators, pagoda-shaped structures, and vacuum degassing units. Owsley (1981) and Bouck et al. (1984) evaluated several aeration systems and selected packed columns for their efficacy in decreasing nitrogen and increasing oxygen. Most packed columns consist of tubes filled with spherical or cylindrical plastic objects to break

the surface tension of the water and release excess gases (Fig. 3). The vertical columns generally contain at least 4 ft of packing material; the diameter of the column is selected to accommodate the volume of water needed. Packed-column aeration is perhaps the least complex and most practical method for treating nitrogen-supersaturated or oxygen-deficient culture water. Like other aeration techniques, however, it does not decrease nitrogen gas supersaturation to 100% or less.

Bouck et al. (1984), who evaluated various types of materials used in packed columns, determined that 2-in. ballast rings were more efficient than other plastic structures. As in any other aeration device, however, increases in water flow and total gas pressure usually decrease efficiency. Consequently, all packed-column aeration systems should be regularly monitored to ensure that they are providing the required level of treatment.

Some mechanical degassing systems reduce gas saturation levels to 100% or less (Fuss 1986). McLaughlin and Busch (1981) demonstrated that the application of a slight vacuum to a packed column caused a sufficient reduction in pressure to release dissolved gases (Fig. 4). This vacuum degassing concept has been adopted at many hatcheries and fishery research laboratories where gas supersaturation cannot be tolerated, as in the culture of particularly sensitive species. In a comparison of the efficiency of packed-column aerators and a vacuum degasser for treating supersaturated waters, packed-column aerators decreased supersaturation from 133% to about 104% and vacuum degassing decreased it to 100% or less (Marking et al. 1983). However, the vacuum degasser also reduced the dissolved oxygen from 27% to 22% saturation, whereas the packed-column aerators increased the oxygen to more than 91% saturation.

Later studies suggested that an integrated system would be more practical (Dawson and Marking 1986). After well water was passed through packed columns, nitrogen gas was reduced from 133% to 105% saturation. When this water was then subjected to a vacuum pressure of only 3 in. of mercury, the degasser further reduced the nitrogen levels to less than 100% saturation. Oxygen was at 86% saturation after treatment with the integrated equipment. The application of aeration before degassing corrected the supersaturation problem and relieved oxygen deficiency problems with little cost and effort.

The latest equipment for treating gas supersaturated water involves the injection of oxygen into water that is held under a slightly negative pressure. The system

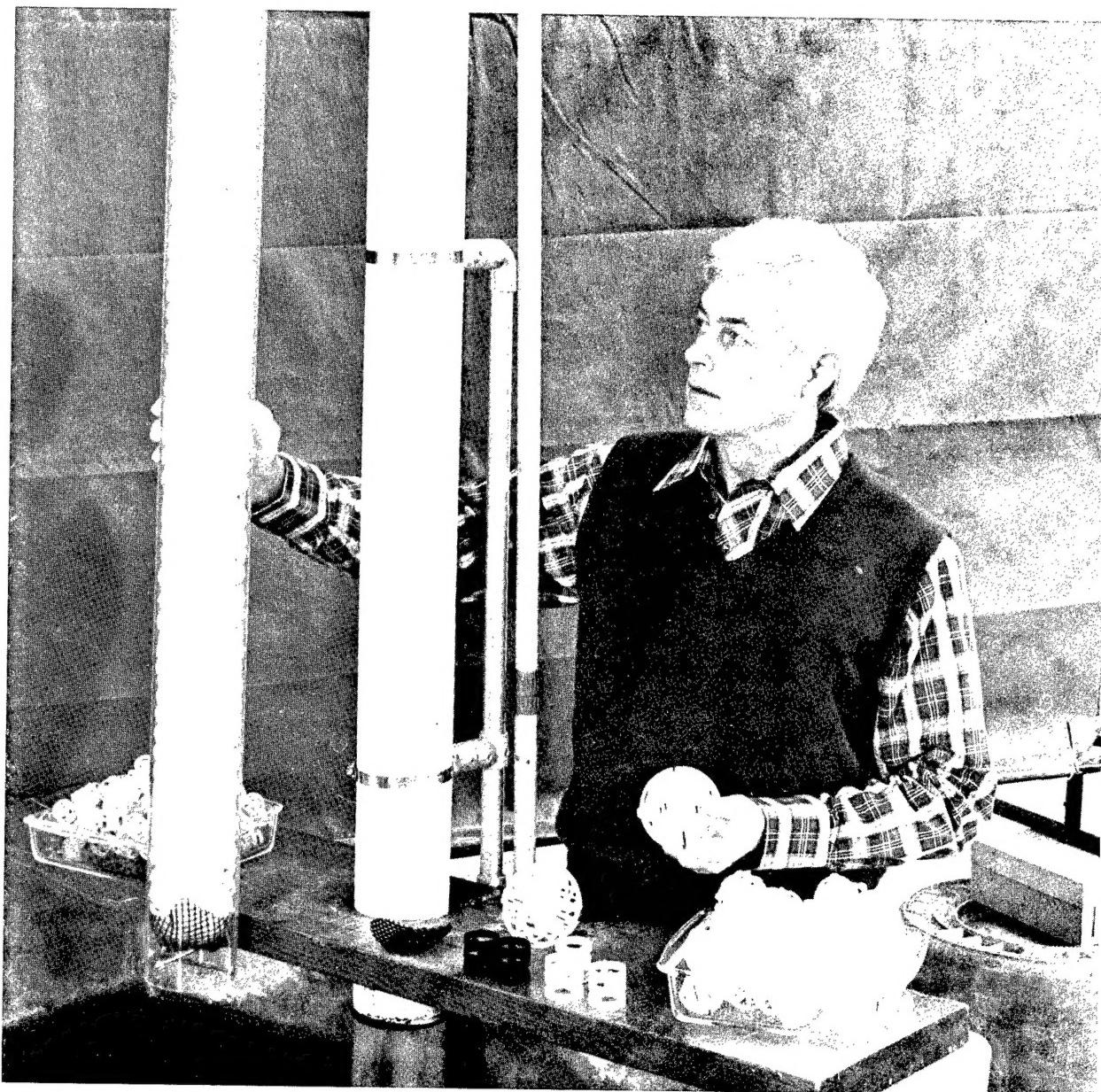


Fig. 3. Packed-column aeration systems effectively increase oxygen and decrease nitrogen gas to about 104% saturation. These columns are filled with commercially available plastic packing materials. The prototype plexiglass column at left is filled with spherical objects, and the opaque plastic column at right (which is in operation) with cylindrical objects.

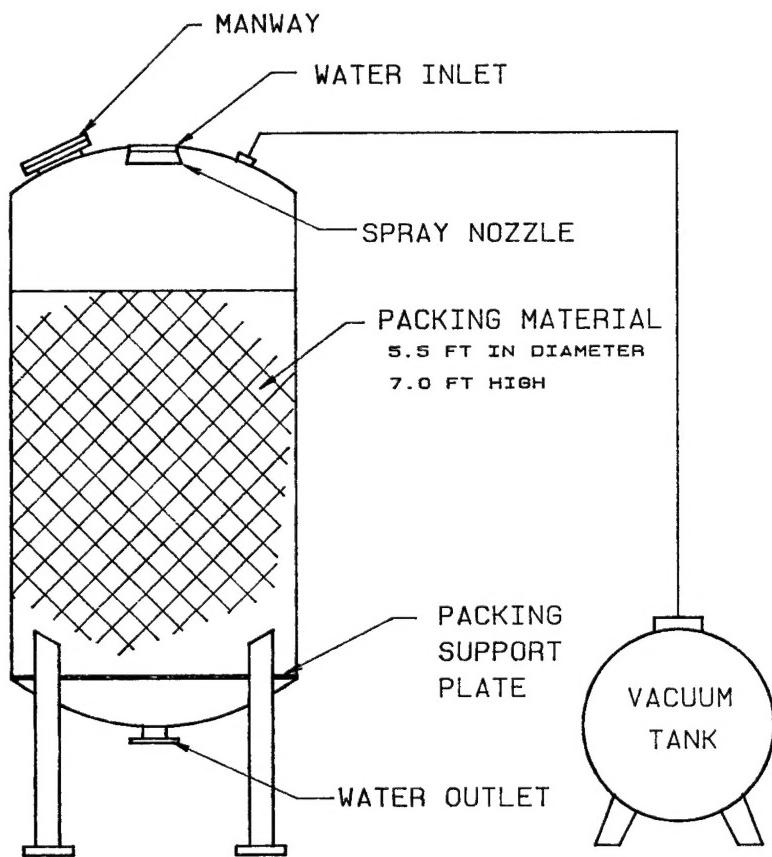


Fig. 4. Vacuum degassing system: the on-line tank contains packing material that provides a large water surface area for release of gases. Sufficient vacuum is applied to the degassing tank to decrease nitrogen and total gas pressure to less than 100% of saturation.

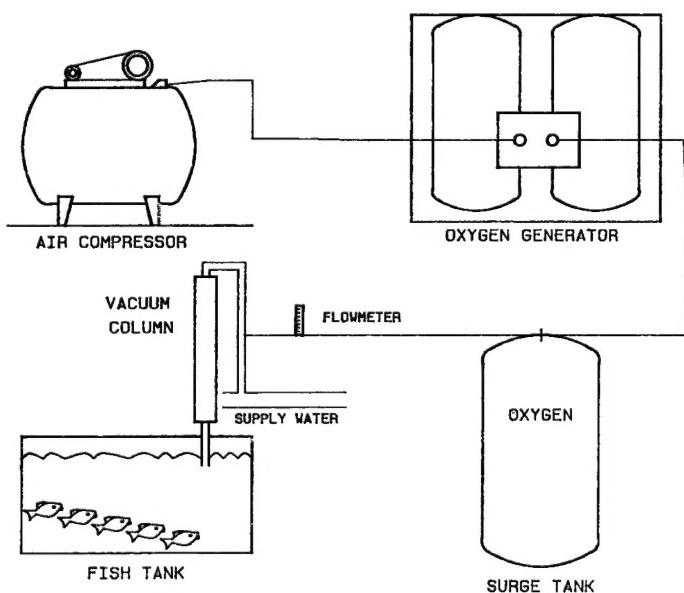


Fig. 5. Typical oxygen injection system for treating gas-supersaturated water consists of an air compressor, an oxygen generator, a surge tank that stores oxygen and maintains pressure, a flowmeter that measures the rate of introduction of oxygen, and a vacuum column that provides a negative pressure.

consists of an air compressor, an oxygen generator, a surge tank for storing oxygen and maintaining pressure, and a vacuum column in which gravity flow of the water produces the needed negative pressure (Fig. 5). Such systems have been in operation at several salmonid hatcheries in Michigan for 2 years or longer.

Oxygen generation systems (Fig. 6) have molecular sieves that adsorb nitrogen and allow oxygen to pass through. When nitrogen loading on a sieve reaches saturation, it can be purged by reducing the pressure. The adsorbed nitrogen can then be drained and released into the air as waste. During nitrogen release by one sieve, the alternate sieve in a unit continues to produce oxygen, thus providing a continuous flow of oxygen to the system.

Oxygen that is injected into the water line effectively scrubs out the excess nitrogen gas under a negative pressure of about 2 in. of mercury. At the same time,

dissolved oxygen increases to 115-120% saturation or even higher, if desired. Because this continuous treatment system markedly improves water quality, equipment costs are offset by improved growth rate and feed conversion efficiency. In a single year of monitoring, use of the system at the Harrietta (Michigan) State Fish Hatchery increased production by about 30%, according to the hatchery manager.

At hatcheries that have installed oxygen injection systems, fish culturists generally agree that fish survival has been increased, fin quality improved, disease incidence decreased, feed conversion improved, and production increased. Although complete details of oxygen generation systems have not been published, the technology is now available and the concepts should be given serious consideration for use in hatchery renovation or new hatchery construction where gas supersaturation is a problem or where increased production is a goal.

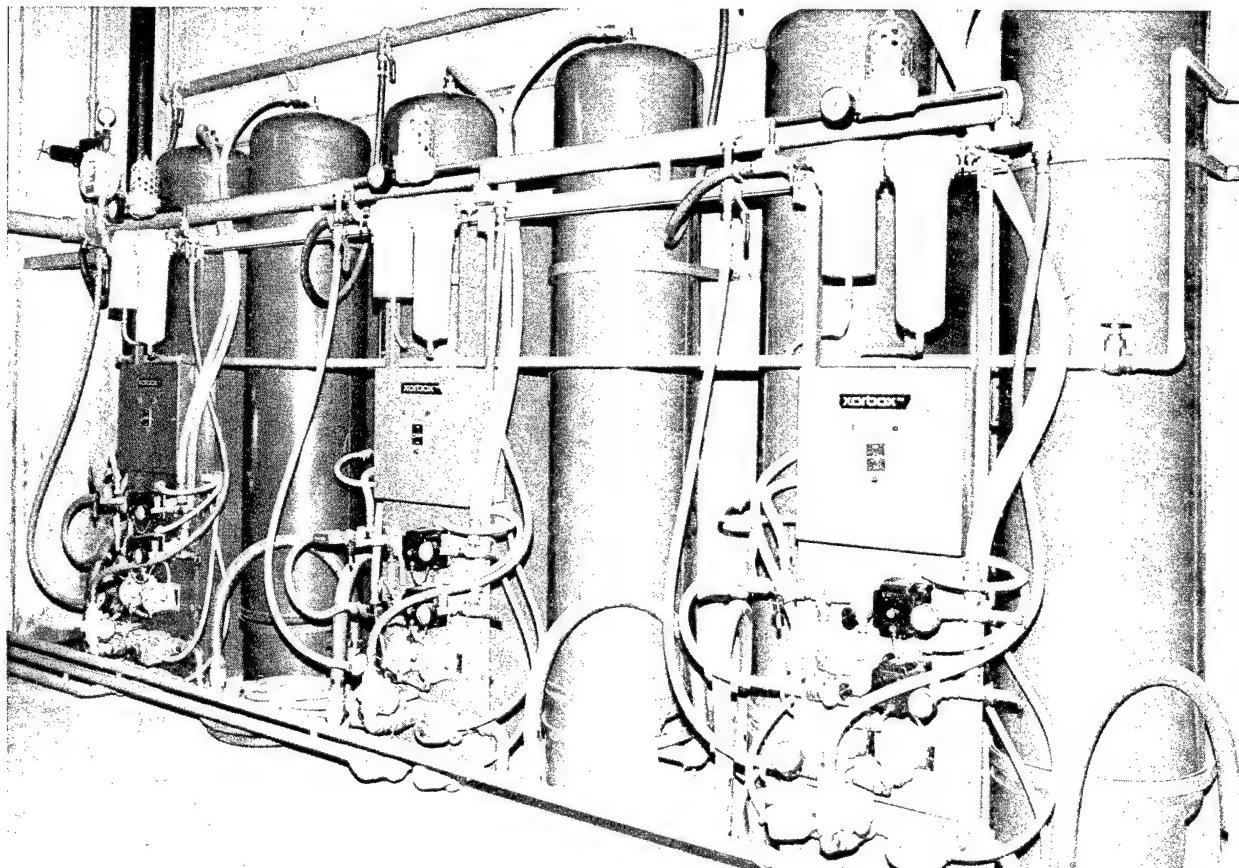


Fig. 6. These oxygen generators are composed of three paired units, each of which produces 200 ft³ of pure oxygen per hour. The units separate and exhaust nitrogen gas from compressed air and discharge pure oxygen into a pressurized air tank for distribution to fish culture and rearing tanks.

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Marking, Leif L. 1987. **Gas supersaturation in fisheries: causes, concerns, and cures.** U.S. Fish Wildl. Serv., Fish Wildl. Leafl. 9. 10 pp.

Millions of cultured and wild fish have been lost to gas bubble disease, which is caused by the supersaturation of air in water. Gas bubbles form in the bloodstream and visibly on external surfaces of fish and in lesions in mouth cavities and eye sockets. Extreme exposure leads to "popeye," disequilibrium, and death. Treatment of gas-supersaturated water by packed-column aeration, vacuum degassing, or oxygen injection alleviates the problem; oxygen injection into hatchery water efficiently removes nitrogen gas and increases dissolved oxygen to levels that may increase fish production.

Key words: Supersaturation, gas bubble disease, freshwater fish, packed-column aeration, vacuum degassers, oxygen injection.

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Marking, Leif L. 1987. **Gas supersaturation in fisheries: causes, concerns, and cures.** U.S. Fish Wildl. Serv., Fish Wildl. Leafl. 9. 10 pp.

Millions of cultured and wild fish have been lost to gas bubble disease, which is caused by the supersaturation of air in water. Gas bubbles form in the bloodstream and visibly on external surfaces of fish and in lesions in mouth cavities and eye sockets. Extreme exposure leads to "popeye," disequilibrium, and death. Treatment of gas-supersaturated water by packed-column aeration, vacuum degassing, or oxygen injection alleviates the problem; oxygen injection into hatchery water efficiently removes nitrogen gas and increases dissolved oxygen to levels that may increase fish production.

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A list of recent *Fish and Wildlife Leaflets* follows.

1. Acid Rain: Effects on Fish and Wildlife, by Kathleen Stecher Mayer, Ell-Piret Multer, and R. Kent Schreiber. 1985. 8 pp.
2. Interpretation of Criteria Commonly Used to Determine Lead Poisoning Problem Areas, by Milton Friend. 1985. 4 pp.
3. Kenai River Salmon . . . A Unique Resource in South-central Alaska, by Carl V. Burger, David B. Wangaard, and Richard L. Wilmot. 1985. 14 pp.
4. Monitoring Fish and Wildlife for Environmental Contaminants: The National Contaminant Bio-monitoring Program, by Joel Jacknow. 1986. 15 pp.
5. Infectious Diseases of Cultured Fishes: Current Perspectives, by G. L. Bullock and Ken Wolf. 1986. 13 pp.
6. Some Parasites and Diseases of Warmwater Fishes, by Glenn L. Hoffman and Andrew J. Mitchell. 1986. 22 pp.
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